autopsy reports were reviewed on all patients who died in the hospital to determine cause of death.

Results: Thirty patients (26%) were pronounced dead on arrival. Of the 82 alive patients, 33 (40%) died in the hospital. The mean injury severity score for patients who died was 43 ± 12 compared with 34 ± 12 for surviving patients (P = .001). In-hospital deaths were caused by aortic rupture in 12 patients (15%), other traumatic injuries in 19 (23%), and multisystem organ failure after aortic repair in two patients (2%). The 12 deaths from aortic rupture occurred ≤4 hours of injury: five died in the emergency department or computed tomography scanner, and seven died in the operating room. Aortic injury was recognized in only two of the seven patients who died intraoperatively and therefore represent the only patients who might have been salvaged with immediate TEVAR. Fifty-nine patients survived >4 hours with contained TAD, and eight (14%) died of associated injuries. Nineteen (32%) underwent aortic repair (17 open, 2 TEVAR) ≤24 hours due to severe aortic disruption or minimal associated injuries. Two died of postoperative complications. Twenty-two patients (37%) had delayed repairs (13 open, 9 TEVAR) because of infectious complications or the need to treat other organ system injuries; all survived to discharge. The mean hospital length of stay after TEVAR was 30 ± 30 days, which was not different compared with 33 ± 27 days after open repair. Operative complications occurred in eight of 30 patients after open repair and in one of 11 after TEVAR (P = NS). Regardless of the repair type, seven of the 19 patients (37%) in this series who underwent repair ≤24 hours developed operative complications compared with two (9%) of the 22 who underwent delayed repair (P = .08). Of the 10 (17%) surviving patients with minimal TAD who were treated with β-blockade and observation, three have stable aortic injuries and seven were lost to follow-up.

Conclusions: Most patients with TAD who arrive alive at the hospital do not experience aortic rupture. Rupture appears to occur within the first 4 hours of admission, before the injury can be recognized in time for salvage with immediate TEVAR. No patient in this series who survived >4 hours died of aortic rupture. Death was related to the extent of associated injuries and was not influenced by the type or timing of repair.

Deglutition Syncope: A Manifestation of Vagal Hyperactivity Following Carotid Endarterectomy

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Background: Transient hypotension and bradycardia after carotid endarterectomy (CEA) is related to increased baroreceptor activity at the carotid bifurcation. These symptoms, mediated by afferent impulses through the nerve of Hering, result from increased vagal activity. We report a case of deglutition syncope after CEA.

Case report: A 61-year-old man presented with left amaurosis fugax and bilateral >80% internal carotid artery stenoses. His surgical history was significant for a right CEA 12 years previously. After an uneventful left CEA, he was hemodynamically stable and without neurologic deficits. On the first postoperative day, the patient experienced crushing chest pain, bradycardia, hypotension, and bilateral vision loss as he began to eat breakfast. Results of an electrocardiogram, computed tomography (CT) scan with pulmonary embolism protocol, cardiac isoenzyme analysis, and echocardiogram were normal. His blood pressure, chest pain, and vision loss responded initially to the administration of pressors. During the next day, similar symptoms developed each time he attempted to eat. With initiation of anticholinergic medication, he was able to eat without symptoms. Results of a barium swallow were normal. The patient's symptoms resolved, and the anticholinergic medication was discontinued. The patient underwent an uneventful right CEA 3 months later. He was hemodynamically and neurologically stable during the procedure and postoperatively. The morning after the CEA, diaphoresis, hypotension, and tachycardia again developed when he began eating breakfast. Anticholinergic medications were again initiated and all symptoms resolved ≤48 hours.

Results: The patient has been seen in follow-up. He has had no further episodes of deglutition syncope or other neurologic symptoms. His CEA are widely patent.

Conclusions: Increased vagal tone after CEA, mediated by hypersensitivity of the baroreceptors of the carotid sinus, is well described. The nerve of Hering, an afferent branch of the glossopharyngeal nerve, synapses with efferent fibers of the vagus nerve in the nucleus tractus solitarius within the medulla. The glossopharyngeal nerve also transmits afferent impulses from the esophagus. We hypothesize that after CEA, our patient had a transient increase in afferent activity to the nucleus tractus solitarius related to changes of the carotid baroreceptor. With additional glossopharyngeal afferent activity associated with swallowing, deglutition syncope developed. Deglutition syncope is considered an unusual manifestation of vasovagal episodes and usually has been reported in association with esophageal, cardiac, and thoracic aortic abnormalities. To our knowledge, this patient is the first case of deglutition syncope reported after CEA. Because deglutition syndrome can be associated with esophageal abnormalities, a barium swallow should be evaluated. Clinicians should be aware of and know how to address this rare complication of CEA.

Late Erosion of a Prophylactic Inferior Vena Cava Filter into the Aorta, Right Renal Artery, and Duodenal Wall

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Background: Prophylactic insertion of inferior vena cava filters (IVCFs) accounts for more than half of all IVCFs placed into multisystem trauma patients with contraindications to anticoagulation. The use of prophylactic IVCFs has increased in association with the introduction of retrievable IVCFs (R-IVCF), most of which are not removed. In this report we describe a patient with R-IVCF–related pseudoaneurysms of the infrarenal aorta and right renal artery who presented 10 months after multiorgan trauma and prophylactic R-IVCF placement. Management required autogenous aortic reconstruction, caval repair, and subsequent right nephrectomy.

Methods: A 42-year-old man presented with left upper extremity edema and was diagnosed with left arm thrombophlebitis. A computed tomography (CT) scan of the abdomen and pelvis was obtained as part of an evaluation of back pain. The CT demonstrated an IVC filter with struts protruding outside the vena cava lumen associated with fluid collections, an infrarenal aortic pseudoaneurysm, and penetration into the duodenal wall (Figs 1 and 2). A follow-up CT scan 3 days later demonstrated increased pericaval fluid collections and new septic emboli to the lungs. The patient's Celect R-IVCF (Cook Medical, Bloomington, Ind) had been placed 10 months earlier for pulmonary embolus prophylaxis in the absence of documented deep vein thrombosis after a motor vehicle accident associated with multiorgan injuries. At the completion of his trauma hospitalization, an attempt to retrieve the filter was unsuccessful and it was left in place. After a second unsuccessful attempt to remove the filter percutaneously, we proceeded with open removal of the R-IVCF requiring retrohepatic caval control, bilateral renal vein control, and infrarenal caval control. One filter strut was imbedded in the duodenal wall but had not entered the lumen. The aortic pseudoaneurysm was repaired using a femoral-popliteal vein interposition graft. The patient was discharged home on postoperative day 20. Follow-up contrast CT demonstrated a right renal artery pseudoaneurysm (Fig 3) that was not present on the preoperative CT. Arteriography demonstrated a pseudoaneurysm arising from the right main renal artery bifurcation at the previous location of one of the filter struts. The patient was not considered a reasonable candidate for renal salvage and was managed with embolization, followed by right nephrectomy.

Results: The patient is doing well at the 3-month follow-up, with normal renal function. He is asymptomatic, with evidence of patency of his IVC and aortic repair.

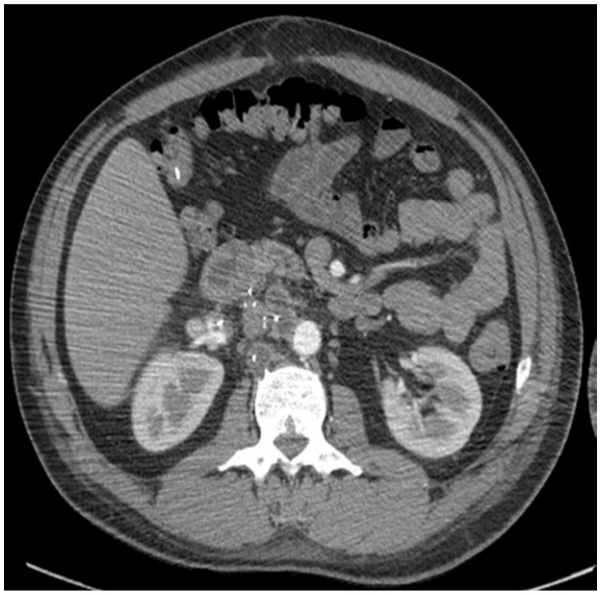

Figure 1: Extraluminal struts from a retrievable IVC filter with associated fluid collections, infrarenal abdominal aortic pseudoaseurysm, and involvement of the duodenal wall.

Fig 1. Computed tomography scan shows extraluminal struts from a retrievable inferior vena cava filter with associated fluid collections, infrarenal abdominal aortic pseudoaneurysm, and involvement of the duodenal wall.

Fig 2. Reformatted 3-dimensional computed tomography scan image of the retrievable inferior vena cava filter shows extraluminal struts and aortic pseudoaneurysm.

Conclusions: The use of prophylactic R-IVCFs after multisystem trauma is increasing; most are not removed. This case report demonstrates that R-IVCFs may be associated with significant risks. We urge caution in the prophylactic use of R-IVCFs until there are data demonstrating the benefit of such a management strategy.

Bilateral Renal Vein Thrombosis Treated with Percutaneous Thrombolysis and Mechanical Thrombectomy

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Background: Renal vein thrombosis is a rare phenomenon with significant morbidity. Management usually consists of systemic anticoagulation, yet with the evolution of endovascular technology, more novel treatment options have been described. We report a case illustrating our approach.

Methods: A 34-year-old previously healthy Caucasian man presented to the emergency department with gradual onset of bilateral flank abdominal pain and anuria. Initial evaluation revealed only proteinuria, but before the initiation of hemodialysis, he underwent an abdominal/pelvic computed tomography (CT) scan. This demonstrated an extensive thrombus involving both renal veins and extending proximally into the juxtarenal inferior vena cava (IVC). After the initiation of heparin, the patient underwent suprarenal IVC filter placement through a transjugular approach. This was followed by IVC venography through a femoral approach, confirming the aforementioned results (Fig), and selective bilateral renal venography starting first with the right renal vein. Pulse spray thrombolysis using tissue plasminogen activator (tPA; 1.5 mg) was performed, followed by mechanical thrombectomy with the AngioJet catheter. Repeat renal venography demonstrated patency. Pulse spray thrombolysis was repeated in a similar fashion for the IVC (3.5 mg tPA) and left renal system (2 mg tPA) with subsequent AngioJet thrombectomy. The final venogram revealed residual clot burden in both renal veins; therefore, separate catheters were selectively placed in both renal veins and treated with continuous tPA infusion at 0.5 mg/h (total, 1 mg/h) for 12 hours in addition to systemic heparinization.

Results: Venography 12 hours later demonstrated excellent flow through both renal systems and the IVC. Heparin therapy was maintained, which was eventually converted to oral anticoagulation. He maintained excellent urine output, and his creatinine levels trended back towards baseline. In addition, his glomerular filtration rate dramatically improved throughout his hospitalization. Renal duplex ultrasound imaging before discharge again demonstrated excellent flow through both renal venous systems and the IVC. His renal function remained stable almost a year after the event, and he has remained dialysis-free.

Conclusions: Percutaneous mechanical thrombectomy and thrombolysis is an alternative therapeutic option for renal vein thrombosis, which can be performed with good technical success and, in this patient, provided successful renal salvage and excellent early clinical outcomes.

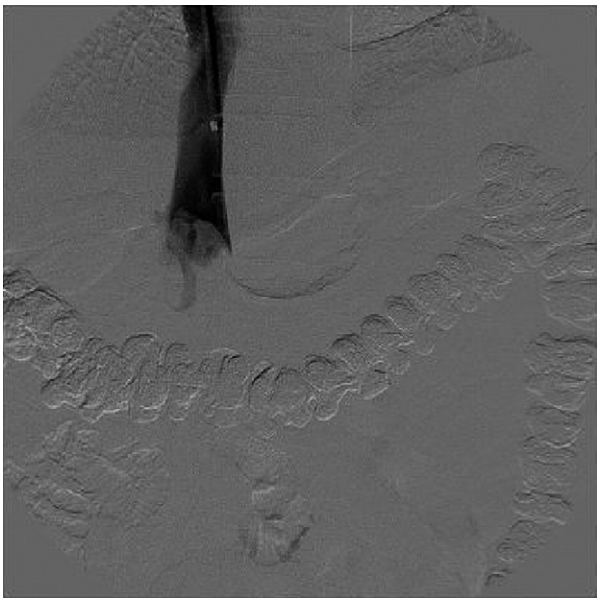

Fig. Venography confirms inferior vena cava filter placement

Management of a Thoracic Aortic Coarctation Presenting as Severe Hypertension and Increasing Dyspnea on Exertion

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Background: Adult thoracic aortic coarctation is universally fatal by the fifth decade if left untreated, primarily due to the accelerated arteriosclerosis. Repair of this rare thoracic pathology in adults presents several technical challenges due to thickness of the aortic wall, difficulties in aortic arch mobilization, large intercostal arteries, and poststenotic aneurysm formation. There is a paucity of discussion of thoracic coarctation in the vascular literature; however, various repair approaches have been advocated in the cardiothoracic literature, including end-to-end anastomosis, path angioplasty repair, and subclavian-to-thoracic aorta bypass.

Methods: A morbidly obese (body mass index, 48 kg/m^2) 35-year-old woman with hypertension, increasing buttock claudication to <50 feet, and dyspnea on exertion was referred to our center for evaluation of a recently found thoracic aortic coarctation. Despite taking four antihypertensive medications, the patient's hypertension was poorly managed. A pressure gradient >110 mm Hg was present between the brachial and femoral arteries (210 vs 100 mm Hg, respectively). Computed tomography angiography (CTA) of the thorax and abdomen demonstrated the coarctation just distal to the left subclavian artery (Fig 1), a small abdominal aorta (<10 mm), and a large cohort of chest and abdominal wall collaterals emanating

Fig 1. Computed tomography demonstrates the coarctation just distal to the left subclavian artery.